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Recommended Citation

Sampson, Tomoko Rie; Dhar, Rajat; and Zipfel, Gregory J., "Cerebral infarction following a seizure in a patient with subarachnoid hemorrhage complicated by delayed cerebral ischemia." *Surgical Neurology International*.2,1. . (2011).
http://digitalcommons.wustl.edu/open_access_pubs/5223

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Case Report

Cerebral infarction following a seizure in a patient with subarachnoid hemorrhage complicated by delayed cerebral ischemia

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Received: 7 October 10

Accepted: 21 November 10

Published: 31 January 2011

Surg Neurol Int 2011, 2:14

This article is available from: <http://www.surgicalneurologyint.com/content/1/2/14>

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This article may be cited as:

SampsonTR, Dhar R, Zipfel GJ. Cerebral infarction following a seizure in a patient with subarachnoid hemorrhage complicated by delayed cerebral ischemia *Surg Neurol Int* 2010;2:14

Available FREE in open access from: <http://www.surgicalneurologyint.com/text.asp? 2011/1/2/76432>

Abstract

Background: Seizures are a recognized complication of subarachnoid hemorrhage (SAH). They can increase the cerebral metabolic demands and lead to cardiopulmonary compromise. This could be detrimental in the setting of delayed cerebral ischemia (DCI), when the brain tissue is vulnerable to further reductions in oxygen delivery or increases in demand. An association between seizures and worsening ischemia could influence the decision to use antiepileptic drug (AED) prophylaxis in patients with vasospasm.

Case Description: A 64-year-old woman developed confusion, aphasia and right hemiparesis on day 7 after aneurysmal SAH. Angiography confirmed severe anterior circulation vasospasm. She initially responded to hypertensive therapy with almost complete resolution of her ischemic neurological deficits. However, on day 10, she had a single generalized seizure and required intubation for airway protection. Her blood pressure dropped with AED initiation, necessitating an increase in the previously stable dose of vasopressors. She developed aphasia and worsening hemiparesis that did not resolve despite hemodynamic augmentation. Subsequent head computed tomographies revealed new infarction in the left anterior cerebral artery territory not present previously. She had received prophylactic phenytoin for only 3 days, as per our SAH protocol.

Conclusion: AED prophylaxis is typically used early after SAH when the risk is high and a seizure may precipitate aneurysmal rebleeding. This case illustrates how a later seizure in the setting of vasospasm can lead to decompensation of DCI, with potential for irreversible infarction. Therefore, patients with vasospasm may benefit from extended durations of prophylaxis to prevent such complications.

Key Words: Anticonvulsants, seizure, subarachnoid hemorrhage, vasospasm

Access this article online

Website:

www.surgicalneurologyint.com

DOI:

10.4103/2152-7806.76432

Quick Response Code:



INTRODUCTION

Seizures may complicate subarachnoid hemorrhage

(SAH) and can precipitate aneurysmal rebleeding, raise intracranial pressure and increase the cerebral metabolic demands. Although seizures most often occur early

after SAH, even prior to hospitalization, patients remain at increased risk throughout their hospital course.^[6] While recent guidelines support the use of antiepileptic drugs (AEDs) for seizure prophylaxis after SAH,^[1] they specifically recommend against long-term administration, as greater AED exposure has been linked to worse outcome.^[5,7] Many institutions, including ours, have now adopted a brief regimen of AED prophylaxis after SAH; one concern regarding this change is that it does not provide coverage during the period when patients are at the highest risk for delayed cerebral ischemia. We present the case of a patient with SAH and clinically stable vasospasm, no longer on AED prophylaxis, who developed cerebral infarction and irreversible neurological deficits after an isolated seizure.

CASE REPORT

A 64-year-old woman with a history of rheumatoid arthritis was diagnosed with SAH based on head computed tomography (CT) after presenting with headache and nausea. The initial neurological examination was normal, but she subsequently developed lethargy due to worsening hydrocephalus, which reversed after a ventriculostomy was placed. Cerebral angiogram revealed a 2.5-mm anterior communicating artery aneurysm that was coiled successfully on day 2 post-SAH. On admission, the patient was loaded with phenytoin (Dilantin) for seizure prophylaxis, and this was continued for the first 3 days of her hospital stay. She remained neurologically stable for the first 6 days of her hospitalization.

On day 7, she developed aphasia with perseveration, dysnomia and disorientation. She also manifested new motor deficits, with right facial weakness and dense right hemiparesis. The head CT was unchanged. Hemodynamic augmentation was immediately initiated, including increased fluid administration and the blood pressure titrated up until a clinical response was noted (mean arterial pressure [MAP] was increased to 110 and then 120–130 mmHg using phenylephrine). An angiogram confirmed severe vasospasm involving the left anterior cerebral artery, which was treated with intra-arterial nicardipine. There was also mild to moderate vasospasm involving the right anterior and bilateral middle cerebral arteries (MCA). Immediately post-procedure, she had persistent dense hemiparesis and aphasia post-procedurally. MAP was further increased and she received a transfusion of packed red blood cells for hemoglobin of 8 g/dl. With maximal hemodynamic therapy (target MAP 140–150 mmHg), her deficits improved with minimal residual facial weakness, antigravity movement on the right side and improved naming. Head CT on day 8 revealed a small new hypodense lesion in the left MCA territory, but this was otherwise unchanged.

She continued on stable doses of vasopressors with

further improvement in her neurological status. By day 10, she was fully oriented, minimally aphasic and had only pronator drift on the right side. However, later that evening, she had a brief tonic-clonic seizure associated with decreased mental status, for which she was intubated. She received a 2-mg dose of lorazepam (Ativan), after which her blood pressure fell to MAPs of 60–70s mmHg for approximately 15 min, necessitating a transient increase in vasopressor requirements. She was subsequently loaded with Fosphenytoin (Cerebyx). Emergent head CT showed evolving infarct in the left parietal lobe but no new lesions [Figure 1]. On reexamination post-ictally, she exhibited worsening of her neurological deficits, with right hemiplegia and impaired comprehension, which persisted despite continued aggressive titration of hemodynamic therapy, including fluids and vasopressors. Her worsened expressive language deficits were more obvious after extubation on day 12. Head CT at that time showed a new large area of mixed hypo- and hyperdensity in the left anterior cerebral artery territory consistent with hemorrhagic infarction [Figure 2]. Vasopressor therapy was continued till day 14 and then weaned off when no neurological improvement was observed. She required a gastrostomy tube for feeding due to persistent dysphagia. She was ultimately discharged to a rehabilitation facility on day 28, with no further seizures on AED therapy. When seen at the 1-month follow-up, she had minimal speech output, a spastic right hemiparesis and cognitive deficits involving memory and attention.

DISCUSSION

The patient included in this case report received a brief (3-day) course of phenytoin for seizure prophylaxis, as was our usual practice. She developed clinical vasospasm that was stabilized with hemodynamic augmentation and her deficits had essentially resolved at the time that she experienced a seizure on day 10. She suffered re-

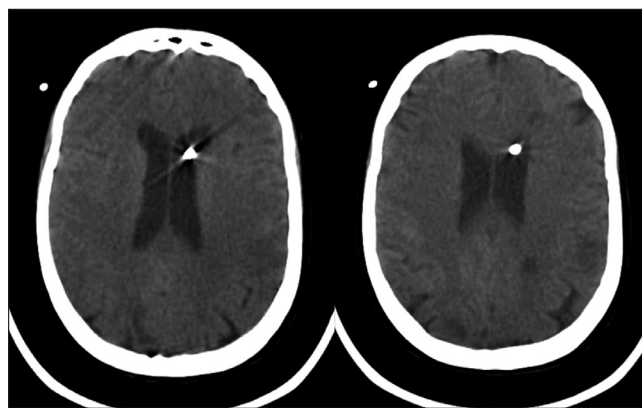


Figure 1: Head computed tomography (day 10) performed soon after her seizure, showing evolution of previously noted hypodensity in the left parietal region

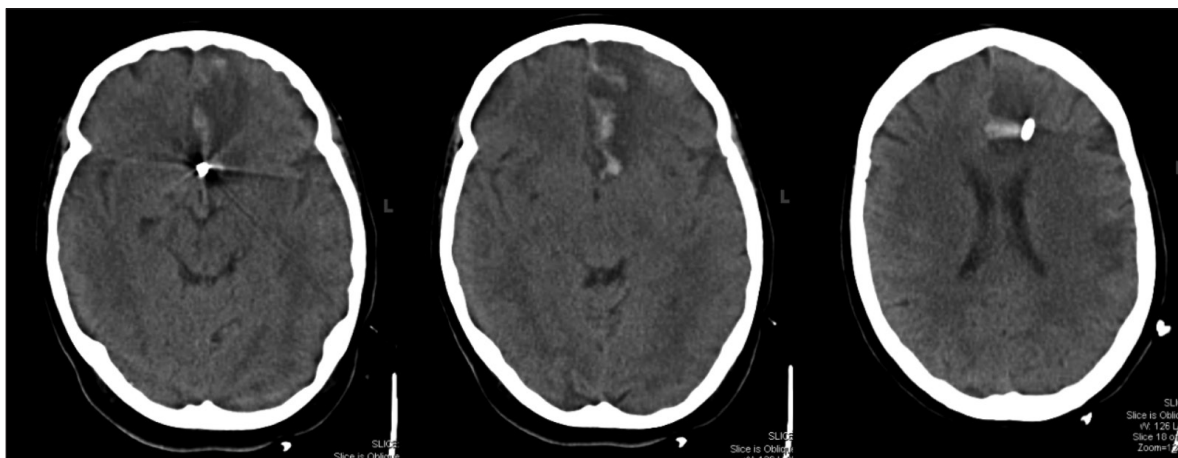


Figure 2: Head computed tomography (day 12) showing new area of infarction with hemorrhagic transformation noted in the left anterior cerebral artery territory

emergent neurological deficits likely related to worsening cerebral ischemia in the territories most affected by angiographic vasospasm. This eventually resulted in radiographic infarction and persistent disability despite maximal medical therapy.

Seizures increase cerebral metabolic demands and can cause neuronal injury.^[3,8] In the setting of significant vasospasm, where cerebral oxygen and glucose delivery is already reduced, an increase in substrate demand could precipitate an acute metabolic crisis, resulting in ischemia. Any hypoxemia and/or hypotension (both seen in our case) that may accompany seizures or their management could also worsen oxygen delivery. The potential for seizures to exacerbate secondary brain injury and raise intracranial pressure has been highlighted in the setting of brain trauma.^[9,10] A similar mechanism may be theorized to explain why an isolated seizure in our patient precipitated irreversible cerebral injury in the setting of vasospasm.

The optimal regimen for AED prophylaxis remains controversial with little to guide formal recommendations.^[4] The potential for seizures to exacerbate delayed cerebral ischemia may influence the duration of seizure prophylaxis after SAH. Many have argued for a shorter course of AEDs after SAH, given the association between their use and worse outcomes.^[5,7] Additionally, one retrospective study found no increase in seizure incidence when comparing a 3-day course of phenytoin to patients receiving longer durations of prophylaxis.^[2] However, as in our case, seizures have been shown to occur in the period when delayed cerebral ischemia is a real concern.^[6] Our case highlights the

potential consequences of epileptic activity in this setting and adds an additional reason to consider a period of AED prophylaxis that extends at least 14 days (i.e., through the peak window of cerebral vasospasm) after SAH. Further studies are needed to better define the optimal length of seizure prophylaxis for this patient population.

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